L Number	Hits	Search Text	DB	Time stamp
1	1	cftr same peptide same open same probability	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/14 14:23
2	27	cftr same peptide same activator	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/14 14:23
3	0	adams-lynn-m.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/14 14:24
5	2	ma-jianjie.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/14 14:25
4	8	davis-pamela-b.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/14 14:25

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PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2


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 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  SEP 09   CA/CAplus records now contain indexing from 1907 to the
                   present
 NEWS  4  DEC 08   INPADOC: Legal Status data reloaded
 NEWS  5  SEP 29   DISSABS now available on STN
 NEWS  6  OCT 10   PCTFULL: Two new display fields added
 NEWS  7  OCT 21   BIOSIS file reloaded and enhanced
 NEWS  8  OCT 28   BIOSIS file segment of TOXCENTER reloaded and enhanced
 NEWS  9  NOV 24   MSDS-CCOHS file reloaded
 NEWS 10  DEC 08   CABA reloaded with left truncation
 NEWS 11  DEC 08   IMS file names changed
 NEWS 12  DEC 09   Experimental property data collected by CAS now available
                   in REGISTRY
 NEWS 13  DEC 09   STN Entry Date available for display in REGISTRY and CA/CAplus
 NEWS 14  DEC 17   DGENE: Two new display fields added
 NEWS 15  DEC 18   BIOTECHNO no longer updated
 NEWS 16  DEC 19   CROPU no longer updated; subscriber discount no longer
                   available
 NEWS 17  DEC 22   Additional INPI reactions and pre-1907 documents added to CAS
                   databases
 NEWS 18  DEC 22   IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields
 NEWS 19  DEC 22   ABI-INFORM now available on STN

 NEWS EXPRESS  DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT
               MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
               AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003
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FILE 'HOME' ENTERED AT 14:31:27 ON 14 JAN 2004

=> file medline biosis embase caplus
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
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	ENTRY	SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 14:31:41 ON 14 JAN 2004

FILE 'BIOSIS' ENTERED AT 14:31:41 ON 14 JAN 2004

FILE 'EMBASE' ENTERED AT 14:31:41 ON 14 JAN 2004

FILE 'CAPLUS' ENTERED AT 14:31:41 ON 14 JAN 2004


```
=> s cftr (p) activat? (p) peptide (p) open (p) probab?
L1            8 CFTR (P) ACTIVAT? (P) PEPTIDE (P) OPEN (P) PROBAB?

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2            2 DUP REM L1 (6 DUPLICATES REMOVED)

=> d l2 total ibib kwic
```

L2 ANSWER 1 OF 2 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER:	2001205320 MEDLINE
DOCUMENT NUMBER:	21107710 PubMed ID: 11158634
TITLE:	Regulation of cystic fibrosis transmembrane conductance regulator single-channel gating by bivalent PDZ-domain-mediated interaction.
COMMENT:	Comment in: Proc Natl Acad Sci U S A. 2001 Jan 30;98(3):787-9
AUTHOR:	Raghuram V; Mak D D; Foskett J K
CORPORATE SOURCE:	Department of Physiology, University of Pennsylvania, Philadelphia, PA 19104-6100, USA.
SOURCE:	PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2001 Jan 30) 98 (3) 1300-5. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY:	United States
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	200104
ENTRY DATE:	Entered STN: 20010417 Last Updated on STN: 20030105 Entered Medline: 20010412

AB The cystic fibrosis transmembrane conductance regulator (**CFTR**) is a cAMP-dependent protein kinase- and ATP-regulated chloride channel, the activity of which determines the rate of electrolyte and fluid transport in a variety of epithelial tissues. Here we describe a mechanism that regulates **CFTR** channel activity, which is mediated by PDZ domains, a family of conserved protein-interaction modules. The Na(+)/H(+) exchanger regulatory factor (NHERF) binds to the cytoplasmic tail of **CFTR** through either of its two PDZ (PDZ1 and PDZ2) domains. A recombinant fragment of NHERF (PDZ1-2) containing the two PDZ domains increases the **open probability** (P(o)) of single **CFTR** channels in excised membrane patches from a lung submucosal gland cell line. Both PDZ domains are required for this functional effect, because **peptides** containing mutations in either domain are unable to increase channel P(o). The concentration dependence of the regulation by the bivalent PDZ1-2 domain is biphasic, i.e., **activating** at lower concentrations and inhibiting at higher concentrations. Furthermore, either PDZ domain alone or together

is without effect on P(o), but either domain can competitively inhibit the PDZ1-2-mediated stimulation of **CFTR**. Our results support a molecular model in which bivalent NHERF PDZ domains regulate channel gating by crosslinking the C-terminal tails in a single dimeric **CFTR** channel, and the magnitude of this regulation is coupled to the stoichiometry of these interactions.

L2 ANSWER 2 OF 2 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2000131261 MEDLINE
DOCUMENT NUMBER: 20131261 PubMed ID: 10666038
TITLE: PKA holoenzyme is functionally coupled to CFTR by AKAPs.
AUTHOR: Huang P; Trotter K; Boucher R C; Milgram S L; Stutts M J
CORPORATE SOURCE: Departments of Medicine and CF/Pulmonary Research and Treatment Center, University of North Carolina, Chapel Hill, North Carolina 27599, USA.. Pingbo_Huang@med.unc.edu
CONTRACT NUMBER: HL-42384 (NHLBI)
HL-533094 (NHLBI)
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY. CELL PHYSIOLOGY, (2000 Feb) 278 (2) C417-22.
Journal code: 100901225. ISSN: 0363-6143.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000427
Last Updated on STN: 20000427
Entered Medline: 20000414

AB Cystic fibrosis transmembrane regulator (**CFTR**) is reported to be preferentially regulated by membrane-bound protein kinase A (PKAII). We tested for close physical and functional association of PKA with **CFTR** in inside-out membrane patches excised from Calu-3 cells. In the presence of MgATP, 8-(4-chlorophenylthio)adenosine 3',5'-cyclic monophosphate (CPT-cAMP) increased the product of **CFTR** channel number and **open probability** (from 0.36 +/- 0.12 to 1.23 +/- 0.57, n = 20, P < 0.0025), and this stimulation was abolished by PKI. Thus Calu-3 membrane isolated from cells retains PKA holoenzyme that is functionally coupled to **CFTR**. PKAII is anchored at specific subcellular sites by A kinase anchoring proteins (AKAPs). Exposure of excised patches to HT-31, a **peptide** that disrupts the association of PKAII and AKAPs, prevented CPT-cAMP stimulation of **CFTR**. Therefore, PKA holoenzyme in isolated membrane patches is bound to AKAPs. In whole cell voltage-clamp studies, intracellular dialysis of Calu-3 cells with HT-31 blocked the **activation** of **CFTR** by extracellular adenosine. These results suggest that AKAPs mediate PKA compartmentalization with **CFTR** and are required for **activation** of **CFTR** by physiological regulators.

```
=> s adams lynn /au
L3            5 ADAMS LYNN

=> s davis pamela /au
L4           17 DAVIS PAMELA

=> s ma jianjie /au
L5          146 MA JIANJIE

=> d his

     (FILE 'HOME' ENTERED AT 14:31:27 ON 14 JAN 2004)

     FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 14:31:41 ON 14 JAN 2004
L1             8 S CFTR (P) ACTIVAT? (P) PEPTIDE (P) OPEN (P) PROBAB?
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L2             2 DUP REM L1 (6 DUPLICATES REMOVED)
L3             5 S ADAMS LYNN /AU
L4            17 S DAVIS PAMELA /AU
L5           146 S MA JIANJIE /AU

=> s cftr (p) activat? (p) peptide
L6           212 CFTR (P) ACTIVAT? (P) PEPTIDE

=> s cftr (p) activator (p) peptide
L7            23 CFTR (P) ACTIVATOR (P) PEPTIDE

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8             7 DUP REM L7 (16 DUPLICATES REMOVED)

=> d l8 total ibib kwic

L8   ANSWER 1 OF 7  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER:   2002:419394  BIOSIS
DOCUMENT NUMBER:    PREV200200419394
TITLE:              Function and specifics of a biological resource including
                    pancreatic cell lines.
AUTHOR(S):          Hay, Robert J. [Reprint author]
CORPORATE SOURCE:   American Type Culture Collection, Manassas, VA, USA
SOURCE:             Proceedings of the American Association for Cancer Research
                    Annual Meeting, (March, 2002) Vol. 43, pp. 1049. print.
                    Meeting Info.: 93rd Annual Meeting of the American
                    Association for Cancer Research. San Francisco, California,
                    USA. April 06-10, 2002.
                    ISSN: 0197-016X.
DOCUMENT TYPE:      Conference; (Meeting)
                    Conference; Abstract; (Meeting Abstract)
LANGUAGE:           English
ENTRY DATE:         Entered STN: 7 Aug 2002
                    Last Updated on STN: 7 Aug 2002
IT   . . .
& Systems of Organisms
        beta cell: endocrine system; liver: digestive system; stomach:
        digestive system
IT   Chemicals & Biochemicals
        ARIP: biomarker; CFTR: expression; CSF-1; activin A;
        azaserine: expression; betacellulin; dexamethasone; gastric mucin:
        secretion; glucagon-like peptide-1 [GLP-1]; glucose; insulin:
        secretion, synthesis; islet duodenal homeobox-1 [IDX-1]: beta-cell
        differentiation factor; plasminogen activator

L8   ANSWER 2 OF 7     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:   2001434728     MEDLINE
DOCUMENT NUMBER:    21212873   PubMed ID: 11316265
TITLE:              Differences in the protein-kinase-A-dependent regulation of
                    CFTR Cl- channels and Na+-K+ pumps in guinea-pig
                    ventricular myocytes.
AUTHOR:             Kockskamper J; Sendhoff K; Erlenkamp S; Bordusa F; Cerovsky
                    V; Glitsch H G
CORPORATE SOURCE:   Loyola University Chicago, Stritch School of Medicine,
                    Department of Physiology, Maywood, IL 60153, USA..
                    jkocksk@luc.edu
SOURCE:             PFLUGERS ARCHIV. EUROPEAN JOURNAL OF PHYSIOLOGY, (2001 Mar)
                    441 (6) 807-15.
                    Journal code: 0154720. ISSN: 0031-6768.
PUB. COUNTRY:       Germany: Germany, Federal Republic of
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
```

ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010806
Last Updated on STN: 20010806
Entered Medline: 20010802

AB Protein-kinase-A- (PKA-) dependent regulation of cystic fibrosis transmembrane conductance regulator (**CFTR**) Cl- current (I(**CFTR**)) and Na+-K+ pump current (Ip) was studied in single guinea-pig ventricular myocytes. Both currents were measured simultaneously by means of whole-cell recording at 30 degrees C. The adenylyl cyclase **activator** forskolin was used to stimulate PKA activity. At -20 mV, forskolin (4 microM) induced a fast activation of I(**CFTR**) and a delayed stimulation of Ip. Despite the strikingly different time courses, however, the potency of the drug to regulate both currents was identical. Half-maximal activation of I(**CFTR**) and stimulation of Ip, respectively, were observed at 9.6 x 10(-8) M and 9.9 x 10(-8) M forskolin. Inclusion of a specific **peptide** inhibitor of PKA in the pipette solution (PKI, 20 microM) blocked forskolin's effect on Ip. However, regardless of the time allowed for cell dialysis, there still was a marked, transient activation of I(**CFTR**), which could be prevented by: (1) a short pre-activation of I(**CFTR**) with forskolin or (2) the additional inclusion in the pipette solution of a synthetic **peptide** (Ht31 **peptide**, 60 microM) that interferes with PKA binding to its anchoring proteins. Thus, there is a tight functional coupling between PKA and **CFTR** Cl- channels in guinea-pig ventricular myocytes. The coupling is probably due to the close physical proximity of channels and kinases. . .

L8 ANSWER 3 OF 7 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2002018354 MEDLINE
DOCUMENT NUMBER: 21338101 PubMed ID: 11444148
TITLE: Guanylin in the human pancreas: a novel luminocrine regulatory pathway of electrolyte secretion via cGMP and CFTR in the ductal system.
AUTHOR: Kulaksiz H; Schmid A; Honscheid M; Eissele R; Klempnauer J; Cetin Y
CORPORATE SOURCE: Department of Molecular Cell Biology, Institute of Anatomy and Cell Biology, Philipps University, Robert-Koch-Strasse 6, 35033 Marburg, Germany.
SOURCE: HISTOCHEMISTRY AND CELL BIOLOGY, (2001 Feb) 115 (2) 131-45. Journal code: 9506663. ISSN: 0948-6143.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20020121
Last Updated on STN: 20020121
Entered Medline: 20011204

AB Cystic fibrosis transmembrane conductance regulator (**CFTR**) is a channel and regulator protein that is crucially involved in transepithelial ion transport. In the exocrine pancreas, the **CFTR** -mediated secretion of an electrolyte-rich fluid is a major but as yet incompletely understood function. We show here that the **peptide** guanylin is a specific **activator** of **CFTR** function in the human pancreas implicating regulation of pancreatic electrolyte secretion. Guanylin and its affiliated signaling and effector proteins including guanylate cyclase C, cGMP-dependent protein kinase II, **CFTR**, and the epithelial Cl-/HCO3- exchanger, anion exchanger 2, are highly expressed in the human pancreas. Guanylin is localized specifically to. . . pathway of electrolyte secretion in the ductal system. Functional studies in two different human pancreatic duct cell lines expressing the **CFTR** Cl- channel that is functionally intact in CAPAN-1 cells but defective (delta F508) in CFPAC-1 cells clearly identify guanylin as a specific regulator of pancreatic

CFTR channel function. Whole-cell patch-clamp recordings in CAPAN-1 cells revealed that forskolin induces an increase of Cl- conductance mediated by cAMP.. . . we conclude that guanylin is an intrinsic pancreatic regulator of Cl- current activation in pancreatic duct cells via cGMP and **CFTR**. Remarkably, in the pancreas guanylin may exert its function through an intriguing luminocrine mode via the pancreatic juice.

L8 ANSWER 4 OF 7 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 1999137573 MEDLINE
DOCUMENT NUMBER: 99137573 PubMed ID: 9950772
TITLE: Cloning, characterization, and functional expression of a CNP receptor regulating CFTR in the shark rectal gland.
AUTHOR: Aller S G; Lombardo I D; Bhanot S; Forrest J N Jr
CORPORATE SOURCE: Department of Medicine, Yale University School of Medicine, New Haven, Connecticut 06510; and Mount Desert Island Biological Laboratory, Salisbury Cove, Maine 04672, USA.
CONTRACT NUMBER: DK-34208 (NIDDK)
P30-ES-3828 (NIEHS)
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY, (1999 Feb) 276 (2 Pt 1) C442-9.
Journal code: 0370511. ISSN: 0002-9513.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF054285
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990413
Last Updated on STN: 19990413
Entered Medline: 19990330
AB In the shark, C-type natriuretic **peptide** (CNP) is the only cardiac natriuretic hormone identified and is a potent **activator** of Cl- secretion in the rectal gland, an epithelial organ of this species that contains cystic fibrosis transmembrane conductance regulator (**CFTR**) Cl- channels. We have cloned an ancestral CNP receptor (NPR-B) from the shark rectal gland that has an overall amino. . . but lacks a glycosylation site and a Glu residue previously considered important for CNP binding. When shark NPR-B and human **CFTR** were coexpressed in Xenopus oocytes, CNP increased the cGMP content of oocytes (EC50 12 nM) and activated **CFTR** Cl- channels (EC50 8 nM). Oocyte cGMP increased 36-fold (from 0.11 +/- 0.03 to 4.03 +/- 0.45 pmol/oocyte) and Cl-. . . +/- 14 to -1,226 +/- 151 nA) in the presence of 50 nM CNP. These findings identify the specific natriuretic **peptide** receptor responsible for Cl- secretion in the shark rectal gland and provide the first evidence for activation of **CFTR** Cl- channels by a cloned NPR-B receptor.

L8 ANSWER 5 OF 7 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 97246495 MEDLINE
DOCUMENT NUMBER: 97246495 PubMed ID: 9115758
TITLE: C-type natriuretic peptide increases chloride permeability in normal and cystic fibrosis airway cells.
AUTHOR: Kelley T J; Al-Nakkash L; Drumm M L
CORPORATE SOURCE: Department of Pediatrics, Willard Bernbaum Cystic Fibrosis Center, Case Western Reserve University, Cleveland, Ohio 44106-4948, USA.
CONTRACT NUMBER: HL-50160 (NHLBI)
P30 DK27651 (NIDDK)
T32 HL07451 (NHLBI)
SOURCE: AMERICAN JOURNAL OF RESPIRATORY CELL AND MOLECULAR BIOLOGY, (1997 Apr) 16 (4) 464-70.
Journal code: 8917225. ISSN: 1044-1549.
PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199704
ENTRY DATE: Entered STN: 19970506
Last Updated on STN: 20000303
Entered Medline: 19970423

AB C-type natriuretic **peptide** (CNP), a hormone which stimulates particulate guanylate cyclase activity, was studied for its ability to stimulate chloride permeability through the cystic fibrosis transmembrane conductance regulator (**CFTR**) in airway epithelial cells. Two cell lines, Calu-3 and CF-T43, were used as models of normal and cystic fibrosis (CF) airway epithelial cells, respectively. Calu-3 cells, derived from a lung carcinoma, express relatively high levels of wild-type **CFTR**. CF-T43 is a transformed line derived from a nasal polyp and expresses the mutant **CFTR**, deltaF508. Calu-3 cells exposed to the nucleotide guanosine-3',5'-monophosphate (cGMP) analogue 8-Br-cGMP exhibit increased 36Cl- efflux, demonstrating that cGMP can mediate. . . Calu-3 monolayers. Whole-cell currents stimulated by CNP display linear current-voltage relationships and have inhibitor pharmacology and ion selectivity consistent with **CFTR** channel activity. Sodium nitroprusside (SNP), an **activator** of soluble guanylate cyclase, and CNP both increase cGMP levels and short circuit current in Calu-3 cells. In contrast, exposure. . . isoproterenol and SNP showed no increase in chloride efflux. Together, these data indicate that CNP can activate wild-type and mutant **CFTR** through a cAMP-dependent protein kinase pathway and that the sensitivity of Calu-3 cells for this stimulation is greater than that. . .

L8 ANSWER 6 OF 7 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 96064750 MEDLINE
DOCUMENT NUMBER: 96064750 PubMed ID: 7592887
TITLE: Isotype-specific activation of cystic fibrosis transmembrane conductance regulator-chloride channels by cGMP-dependent protein kinase II.
AUTHOR: French P J; Bijman J; Edixhoven M; Vaandrager A B; Scholte B J; Lohmann S M; Nairn A C; de Jonge H R
CORPORATE SOURCE: Department of Cell Biology, Faculty of Medicine and Health Sciences, Erasmus University, Rotterdam, The Netherlands.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Nov 3) 270 (44) 26626-31.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199512
ENTRY DATE: Entered STN: 19960124
Last Updated on STN: 19970203
Entered Medline: 19951221

AB . . . alpha cGK (cGKI) purified from bovine lung were compared for their ability to activate the cystic fibrosis transmembrane conductance regulator (**CFTR**)-Cl- channel in excised, inside-out membrane patches from NIH-3T3 fibroblasts and from a rat intestinal cell line (IEC-CF7) stably expressing recombinant **CFTR**. In both cell models, in the presence of cGMP and ATP, cGKII was found to mimic the effect of the catalytic subunit of cAMP-dependent protein kinase (cAK) on opening **CFTR**-Cl-channels, albeit with different kinetics (2-3-min lag time, reduced rate of activation). By contrast, cGKI or a monomeric cGKI catalytic fragment was incapable of opening **CFTR** -Cl- channels and also failed to potentiate cGKII activation of the channels. The cAK activation but not the cGKII activation was blocked by a cAK inhibitor **peptide**. The slow activation by cGKII could not be ascribed to counteracting protein phosphatases, since neither calyculin

A, a potent inhibitor. . . by cGKII closed instantaneously upon removal of ATP and kinase but reopened in the presence of ATP alone. Paradoxically, immunoprecipitated **CFTR** or CF-2, a cloned R domain fragment of **CFTR** (amino acids 645-835) could be phosphorylated to a similar extent with only minor kinetic differences by both isotypes of cGK. Phosphopeptide maps of CF-2 and **CFTR**, however, revealed very subtle differences in site-specificity between the cGK isoforms. These results indicate that cGKII, in contrast to cGKI alpha, is a potential **activator** of chloride transport in **CFTR**-expressing cell types.

L8 ANSWER 7 OF 7 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 92042003 MEDLINE
DOCUMENT NUMBER: 92042003 PubMed ID: 1718974
TITLE: Functional insertion of the SV40 large T oncogene in cystic fibrosis intestinal epithelium. Characterization of CFI-3 cells.
AUTHOR: Chastre E; Di Gioia Y; Barbry P; Simon-Bouy B; Mornet E; Fanen P; Champigny G; Emami S; Gespach C
CORPORATE SOURCE: Institut National de la Sante et de la Recherche Medicale U.55, Unite de Recherches sur les Peptides Neurodigestifs et le Diabete, Paris, France.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1991 Nov 5) 266 (31) 21239-46.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199112
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19960129
Entered Medline: 19911213

AB . . . the human cytokeratin 18 gene and poorly differentiated by phase-contrast and electron microscopy. Functional membrane receptors activated by vasoactive intestinal **peptide** (VIP), its natural analogue pituitary adenylate cyclase activating **peptide** (PACAP-38), and isoproterenol were observed in CFI-3 cells. Restriction fragment length polymorphism analysis of the PstI KM19 site revealed that the **cftr** locus was identical in the chorionic villi and in CFI-3 cells. The manifestation of CF in this family was not. . . by the 125I efflux, was induced in CFI-3 cells by the calcium inophore ionomycin, but not by the adenylate cyclase **activator** forskolin, and was inhibited by the chloride channel blocker 5-nitro-2-(3-phenylpropylamino)benzoic acid. These results were confirmed in patch clamp studies in. . . phenotype relating to defective regulation of Cl-channels, and therefore constitute a suitable model, 1) for elucidating the function of **CFTR** protein, 2) developing new therapeutic agents, and 3) correcting the CF defect by gene replacement therapy in vitro.